Correspondence

The Editorial Board will be pleased to receive and consider for publication correspondence containing information of interest to physicians or commenting on issues of the day. Letters ordinarily should not exceed 600 words, and must be typewritten, double-spaced and submitted in duplicate (the original typescript and one copy). Authors will be given an opportunity to review any substantial editing or abridgement before publication.

Diagnosis of Organophosphate Poisoning

TO THE EDITOR: The diagnosis of mild to moderate organophosphate poisoning can be unequivocal in cases in which one does not have basic cholinesterase values. Midtling and colleagues described in this journal a novel diagnostic approach.[1] They had conducted a back-extrapolation of cholinesterase values to the time of poisoning. Similar cases were recently reported elsewhere.[2]

This method, although very helpful, cannot assist in the diagnosis of acute cases. Therefore, we suggest another simple method of diagnosing organophosphate poisoning: a blood specimen should be drawn for cholinesterase determination on admission of the patient, before and a few minutes after administration of an oxime (such as pralidoxime chloride). An apparent increase of cholinesterase activity after the administration will disclose the diagnosis of organophosphate poisoning, even if the initial levels are within normal limits.

In case oxime therapy has already been given before admission to the emergency room, a comparison should be made between erythrocyte and plasma cholinesterase activity. A markedly decreased cholinesterase activity in the plasma along with normal or near-normal erythrocyte cholinesterase activity will raise the possibility of oxime-treated organophosphate intoxication, since oxime-induced reactivation is much more rapid in erythrocytes than in plasma. This is true in most cases, except for rare cases of decreased plasma cholinesterase activity from other causes.[3]

This reactivation of cholinesterase by oximes is usually possible for a few days following organophosphate intoxications, after which aging of the enzyme occurs. Time for aging varies among various types of organophosphate.

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Catheter-Related Septic Thrombosis

TO THE EDITOR: In their recent review of catheter-related septic central venous thrombosis, Kaufman and co-workers identified 37 previously reported cases that were diagnosed antemortem.[1]

To this number might be added some of the 54 cases reported by Pruitt and colleagues[2] in a reference not cited by Kaufman. In at least 22 of these cases the diagnosis apparently was made antemortem.

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Scuba Diver's Thigh

TO THE EDITOR: Meralgia paresthetica usually is due to compression of the lateral femoral cutaneous nerve at the anterior superior iliac spine. The classical cause is an expanding abdomen secondary to ascites, obesity and pregnancy, but other mechanisms have been described.[1] These include stretching of the nerve by pelvic tilt or standing at attention for long intervals and injury from tight pants,[2] corsets, trusses, a heavy wallet,[3] tourniquets on the thigh[4] and a parachute harness strapped over the groin.[5] Recently we noted a previously unreported cause associated with participation in a popular sport, namely, scuba diving. This case should be of interest to readers and physicians in California who might encounter similar patients.

A 26-year-old woman was scuba diving for approximately 50 minutes at a depth of 80 feet. She wore a 3/8″ full Neoprene II wetsuit, an inflatable buoyancy compensator with tank attached and a webbed belt with 30 lb of free lead weights, used to counteract the positive buoyancy of the wetsuit, that was placed directly on her iliac crests. During the dive she noted a sensation of pressure on her left hip. Upon emerging her left thigh was numb. Six weeks later examination showed sensory loss in the distribution of the left lateral femoral cutaneous nerve. She became asymptomatic over the next four months. Two months later, six months following the initial episode, she again went scuba diving with the same equipment. Immediately thereafter the numbness recurred but subsided in approximately two months. She continues scuba diving but has replaced the original belt that has immovable weights with a shot belt in which the weight is distributed evenly. At present she has no complaints or cutaneous sensory changes.

Our subject, who is 160 cm (63 in) tall, with a 58.5-cm (23-in) waist and a weight of 48.5 kg (107 lb), did not have the abdominal enlargement usually present with meralgia paresthetica. The circumstances leave little doubt that pressure